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SUBJECT Predictions of Cardiovascular Responses During STS Reentry Using Mathematical Models

The physiological adaptation to weightless exposure includes cardiovascular deconditioning arising in part from a loss of total circulating blood volume and resulting in a reduction of orthostatic tolerance. Thus, the crew of the Shuttle orbiter are less tolerant to acceleration forces in the head-to-foot direction during the reentry phase of the flight at a time they must function at a high level of performance. The theoretical study reported herein was undertaken to evaluate ti factors that contribute to orthostatic intolerance during and following reen ry and to predict the likelihood of impaired crew performance. It utilizes a computer simulation approach employing a mathematical model of the cardiovascular system. The results show that, depending on the severity of blood volume loss, the reentry acceleration stress may be detrimental to physiologic function and may place the physiologic status of the crew near the borderline of some type of impairment. They are in agreement with conclusions from early ground-based experiments as well as from observations of early Shuttle flights.

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PREDICTIONS OF CARDIOVASCULAR RESPONSES DURING STS REENTRY USING MATHEMATICAL MODELS

Prepared for
National Aeronautics & Space Administration
Lyndon B. Johnson Space Center
Houston, Texas

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Houston, Texas

September 1984

PREDICTIONS OF CARDIOVASCULAR RESPONSES DURING

STS REENTRY USING MATHEMATICAL MODELS

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ABSTRACT

The physiological adaptation to weightless exposure includes cardiovascular deconditioning arising in part from a loss of total circulating blood volume and resulting in a reduction of orthostatic tolerance. Thus, the crew of the Shuttle orbiter are less tolerant to acceleration forces in the head-to-foot direction during the reentry phase of the flight at a time they must function at a high level of performance. The theoretical study reported herein was undertaken to evaluate the factors that contribute to orthostatic intolerance during and following reentry and to predict the likelihood of impaired crew performance. It utilizes a computer simulation approach employing a mathematical model of the cardiovascular system. The results show that, depending on the severity of blood volume loss, the reentry acceleration stress may be detrimental to physiologic function and may place the physiologic status of the crew near the borderline of some type of impairment. They are in agreement with conclusions from early ground-based experiments as well as from observations of early Shuttle flights.

PREDICTIONS OF CARDIOVASCULAR RESPONSES DURING STS REENTRY USING MATHEMATICAL MODELS*

INTRODUCTION

When the crew of the Shuttle orbiter return at the end of their mission, they are exposed during reentry to acceleration forces in the head-to-foot direction ($+G_z$). This is in contrast to all previous manned space flights in which the crew was exposed only to the more benign transverse forces in the front-to-back direction ($+G_x$) during reentry. Headward acceleration ($+G_z$) tends to pool fluid in the legs. In addition, the crew have undergone a certain degree of cardiovascular deconditioning from their stay in space including a loss of blood volume. Further, their reentry maneuvers involving banking turns produce acceleration forces+ up to twice the normal value due to gravity at sea level. Because of these various factors, namely, the upright sitting posture, loss of blood volume, and high g-forces, the crew are in a physiological state that lends itself to some degree of orthostatic intolerance during reentry and during a time when they must function at a high level of performance. Early ground-based experiments, including bed rest and centrifugation studies, anticipated that despite the small peak g-forces involved, some crewmembers would experience orthostatic intolerance and impaired performance (5,8,17).

Because of this potential problem the shuttle crew is required to wear anti-g garments which they would inflate at the first signs of difficulty. Some of the crew would also use fluid loading as a preventive measure during later flights. They are monitored by electrocardiogram (ECG) during reentry and are administered a stand test soon after landing to assess the cardiovascular status. During the early Shuttle flights of short duration, reentry was accompanied by signs of potential clinical significance (1). It is, therefore, desirable to assess and perhaps predict cardiovascular status during reentry following even longer missions and in a larger crew population who have been deconditioned by space flight.

The objective of the present study was to evaluate, using mathematical models and a computer simulation approach, the factors that contribute to orthostatic intolerance during and following reentry and to predict the likelihood of impaired crew performance. Although mathematical models have been used previously to assist in designing experiments and interpreting results for the Life Sciences Flight Experiment Program (16,24), this is the first attempt to use models to analyze a problem concerning astronaut health and safety. The study, summarized herein, represents a preliminary assessment of the overall problem that was conducted soon after the first STS flight.

* Results of this study have been presented at previous meetings (13,23) and have been included in the STS-1 Medical Report (1).

+ We will henceforth refer to them as g-forces, since they are measured in units of g, the acceleration due to gravity at sea level.

THE MATHEMATICAL MODEL

A mathematical model of the cardiovascular system which was developed for the analysis of Skylab data and validated for lower body negative pressure (LBNP) and tilt responses, was utilized (3) in this study. The model consists of 28 vascular compartments without intravascular leaks and is intended to provide pulsatile responses to short-duration stresses. Figure 1 shows an earlier version of the model, containing only 20 compartments. Each compartment is described by equations which relate transmural pressure, blood flow, and blood volume. The control system for the circulation consists essentially of a baroreceptor system which regulates heart rate, strength of heart contraction, and resistances and compliances of the vessels (Figure 2). The model accounts for the effects of gravity in terms of hydrostatic gradients in each of the compartments. The pressures in the compartments for carotid artery and the upper thoracic aorta represent respectively, pressures at the head and the heart levels, both critical regions involved with orthostatic competency.

The model is well suited to study the cardiovascular effects due to changes in the hydrostatic gradients of the blood columns elicited by $+G_z$ loading. These effects include increased leg pooling, decreased head blood pressure, and cardiovascular reflex responses, all of which are considered in the model.

MODEL VALIDATION STUDIES

The model was originally developed to simulate exercise, lower body negative pressure and tilt table experiments (2,3). It had not previously been validated for conditions that included g-forces higher than 1-g. Therefore, it was first necessary to validate the model for cardiovascular responses over a range of g-forces. Data available from centrifugation studies were the most appropriate for this purpose. Two kinds of data from centrifugation studies in humans were considered, those related to the steady-state behavior and those which measured the dynamic behavior. The approach used was to simulate the experimental conditions and protocols and then compare the model and experimental responses. In most cases the observations were on standard physiological variables such as blood pressures and heart rate. In one case, however, the model was compared to clinical manifestations of tolerance to acceleration stress such as vision impairment. The results of the validation study are discussed below.

A) STEADY-STATE BEHAVIOR

Experimental studies by Gauer (4) and Lindberg and Wood (18) and in the early 60's gathered extensive cardiovascular data from normal subjects in centrifuges at various levels of g-loading. Representative data are shown in Figure 3 by circles and dashed lines. The corresponding steady-state responses of the model are shown by the solid lines. Increasing positive headward acceleration results in significant changes in cardiovascular function. In particular, blood pressures below the hydrostatic indifference point (HIP, located slightly below the level of the heart) tend to rise and blood pressures above the HIP tend to fall. Also, the stroke volume decreases to a greater degree than the heart rate increase resulting in a cardiac output

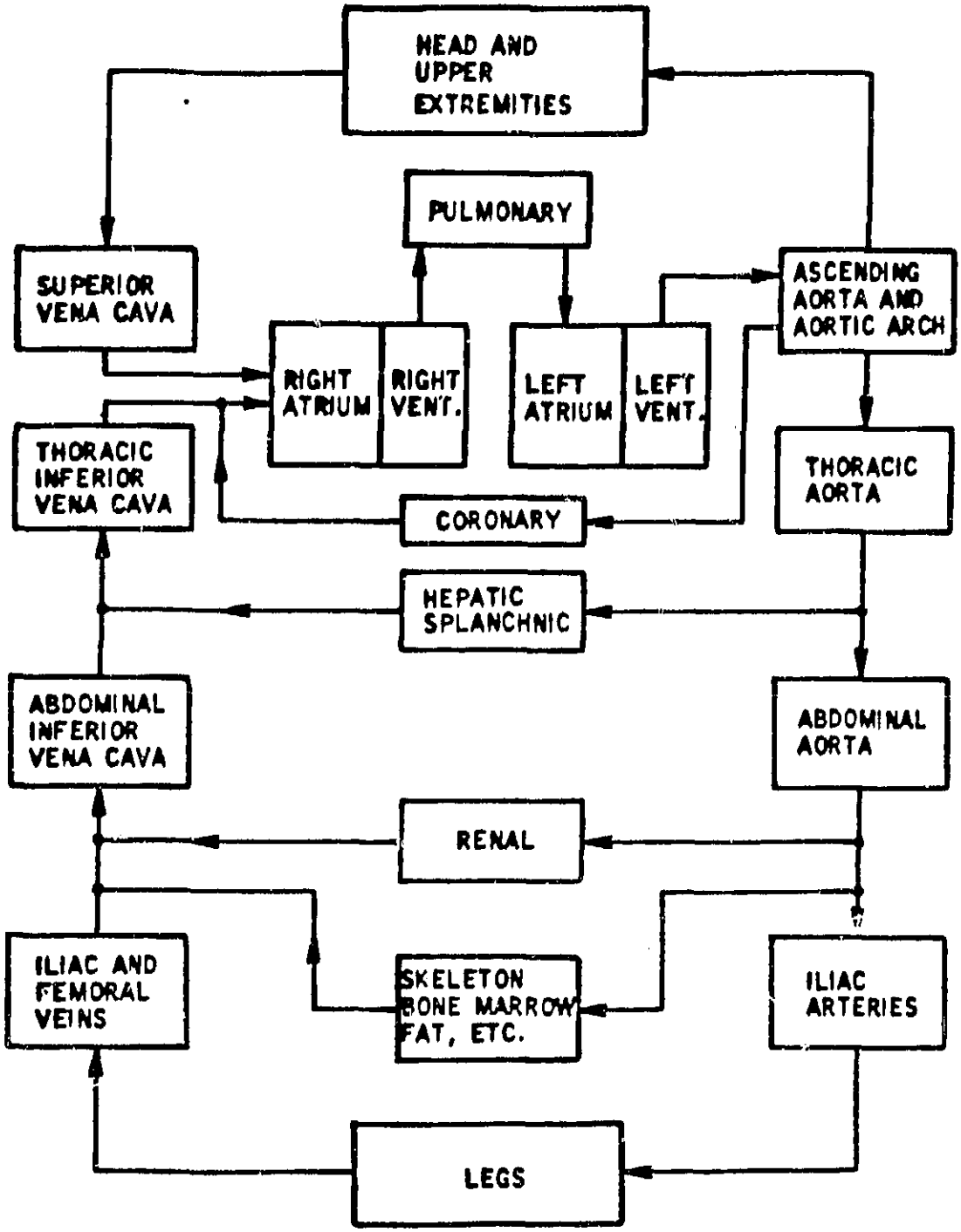


Figure 1: Block diagram of the pulsatile cardiovascular model

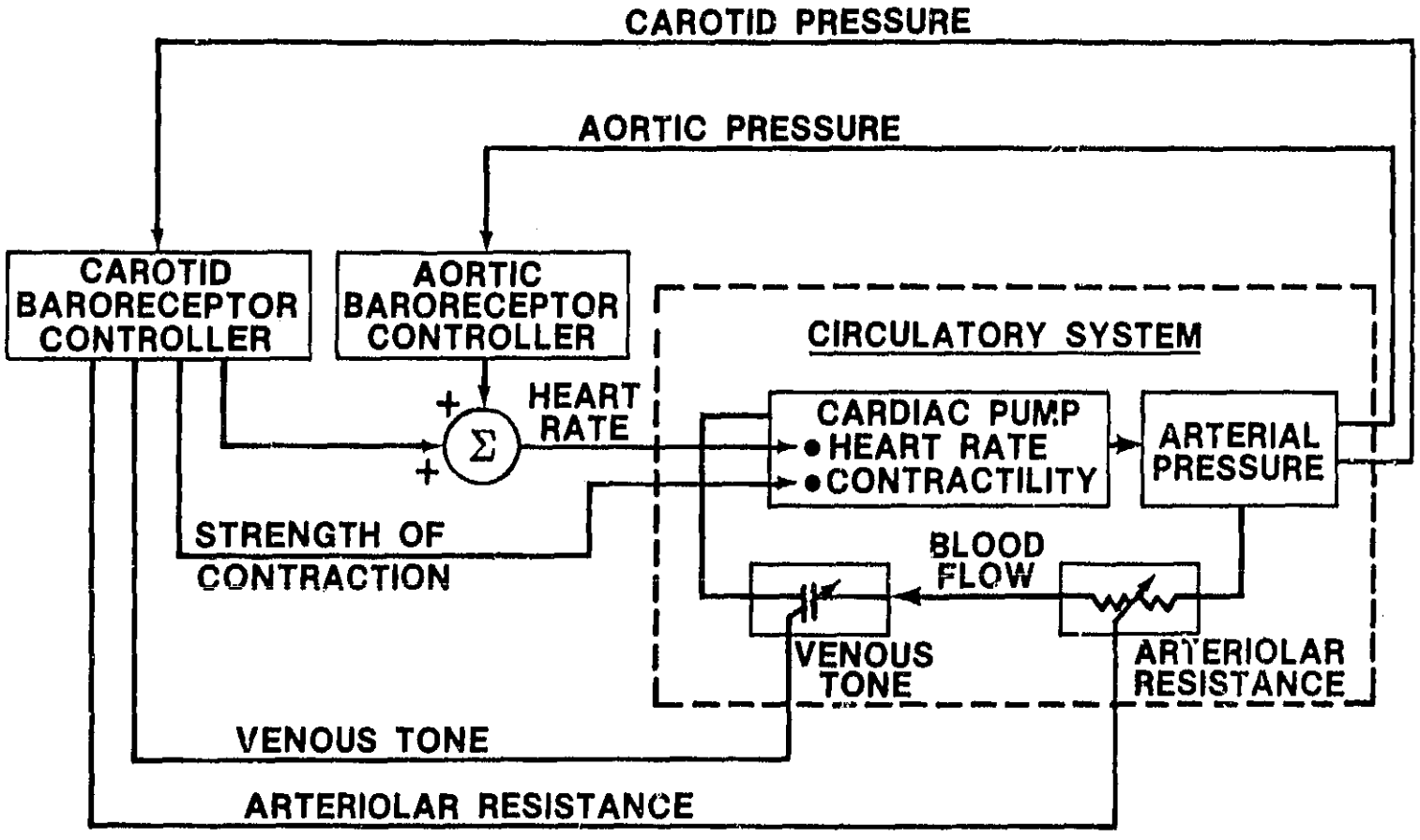


Figure 2: Block diagram of the controlling system associated with the cardiovascular model

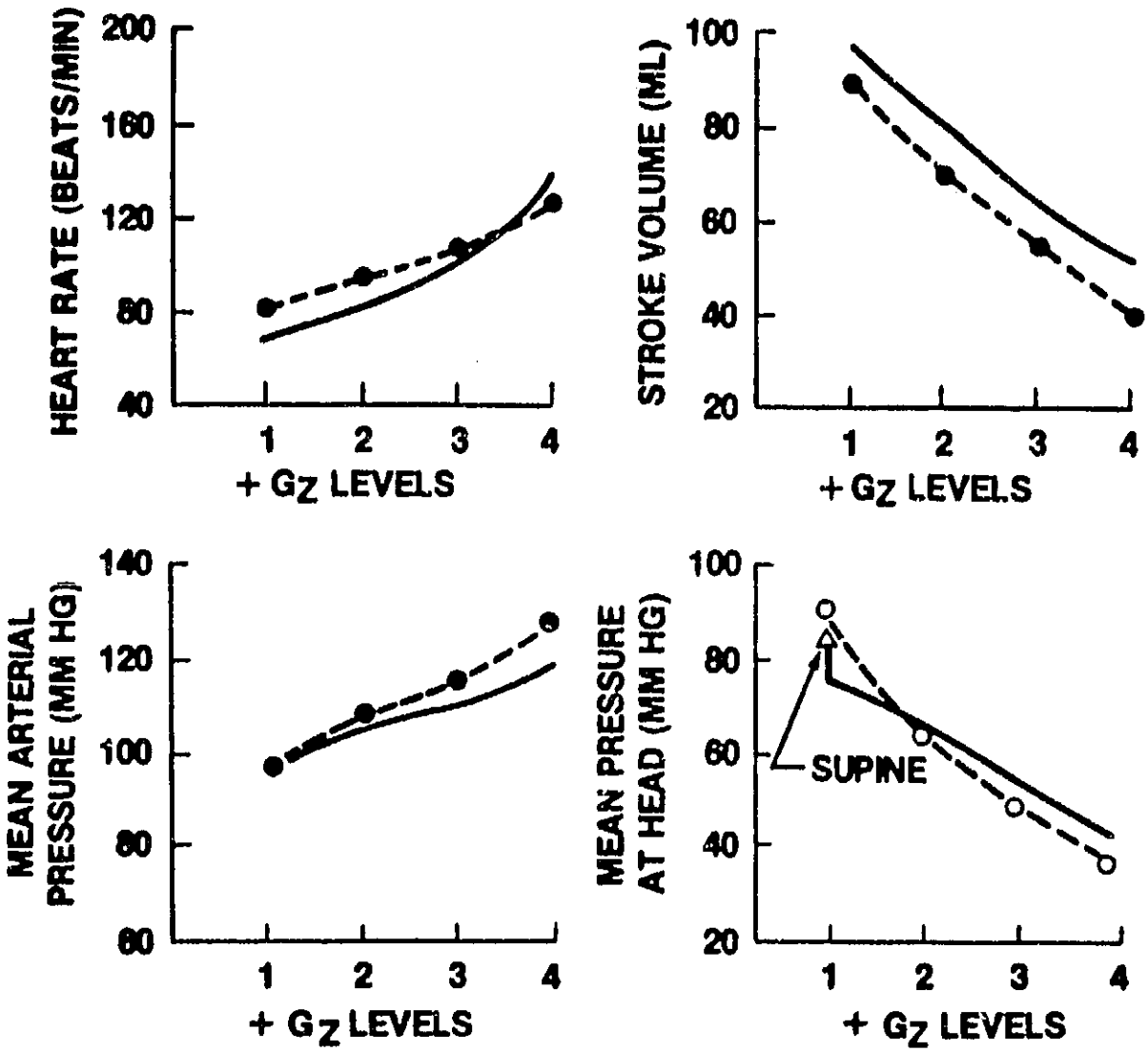


Figure 3: Model response to $+G_z$ acceleration:
heart rate, stroke volume, and arterial pressure

●---● Lindberg and Wood (18)

o---o Henry et al. (7)

——— Simulation

decrement of a maximum of 20-25 percent. The model responses demonstrate these qualitative characteristics and agree quite favorably with the quantitative observations in humans over a wide range of g-forces.

Although agreement is good between model and experimental data for stroke volume, heart rate and blood pressures, there is relatively poor quantitative agreement with the results for cardiac output (CO) and total peripheral resistance (TPR). As indicated in Figure 4, the data of Lindberg and Wood demonstrate a much larger decrease in CO and a much greater increase in TPR than obtained in the modeling studies. It should be pointed out that CO and TPR are closely related physiologically, and the model's small decrease in CO response corresponds to the small increase in TPR. While the discrepancy between model and experimental data on CO and TPR cannot be completely explained, it should be mentioned that in a recent study by Logan (19) similar to the one by Lindberg and Wood (18), CO was found not to decrease, but actually increase by 14 percent at $+G_z$ of 4 g (Figure 4). The increases in heart rate were much more proportionate to stroke volume decreases, in this case. It is not possible to say at the present which of the two experimental studies, Lindberg and Wood's (18) or Logan's (19), accurately reflects the normal CO and TPR responses to g-loading. The variability among subjects and the degree of training seem to be major factors.

B) DYNAMIC BEHAVIOR

Two separate, but similar, experimental studies provided the data needed to verify dynamic behavior of the model. In both these studies, the headward g-force was raised rapidly to 4.5 g and maintained at that level for about 15 seconds before returning to normal one-g. The data are depicted in Figure 5. With increasing g-force, blood is forced away from the heart, and pressure measured at the head level decreases rapidly to about zero. At pressure levels this low, the probability of the subject blacking out is very high. Reflex action of the baroreceptor pathways, however, acts very quickly to restore the pressure, at least partially, toward normal levels. In one study (Lindberg and Wood) the pressure was measured at both the head and the heart level. It can be seen that as pressure at the head level falls, arterial pressure at the heart level rises. This latter elevation in blood pressure is undoubtedly due to baroreceptor reflex action and also due to the fact that the measured pressure is just below the $HIP_{\#}$ the point below which pressures will increase during standing.

The mathematical simulation of this experiment is shown in Figure 6, and demonstrates the model's capability to simulate blood pressure waveforms. The top tracing is the pressure at the head level and the bottom is that at the heart level. These two pressure responses are quite different, as confirmed by measurements on human subjects. The pulsatile characteristics of the model can be seen in this figure, including peak systolic and minimal diastolic pressures. It should be emphasized that these responses are not a result of using empirical equations, but rather, they are based solely on physical principles and general physiological concepts concerning pulsatile flow in a closed elastic system. The model's responses are realistic and demonstrate also the baroreflex effects during the centrifugation simulation. In comparing model and human responses, it is worth noting that the model has realistically reproduced the following events:

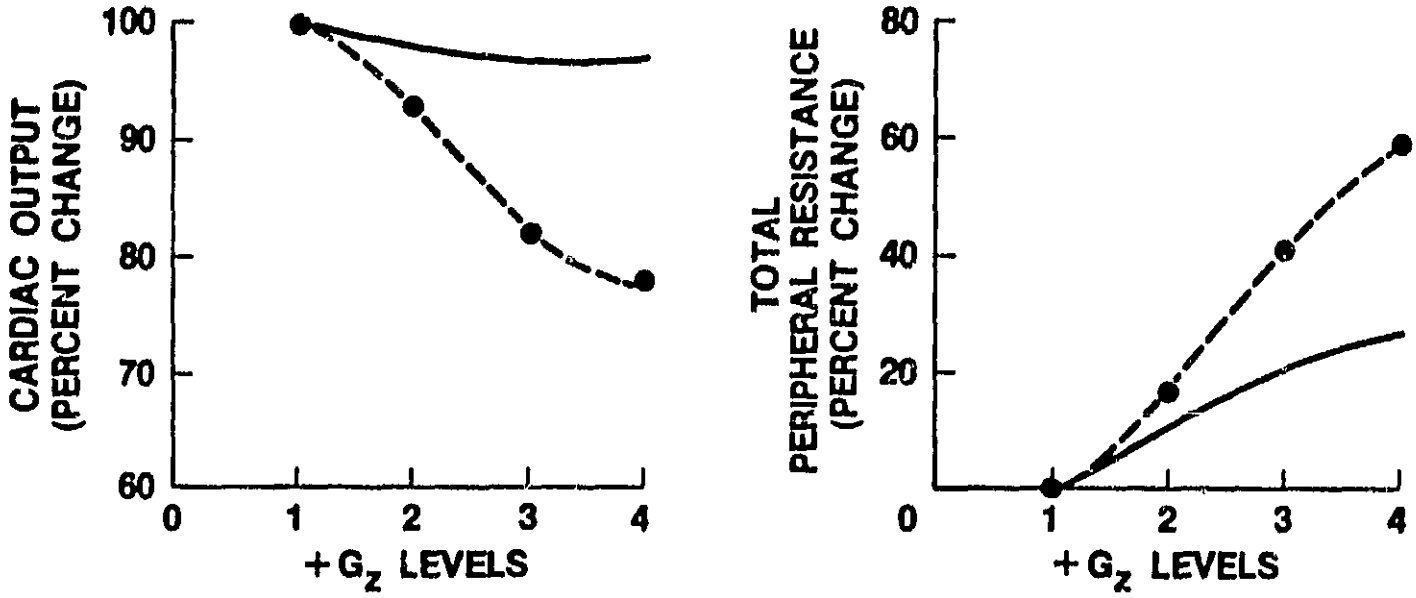
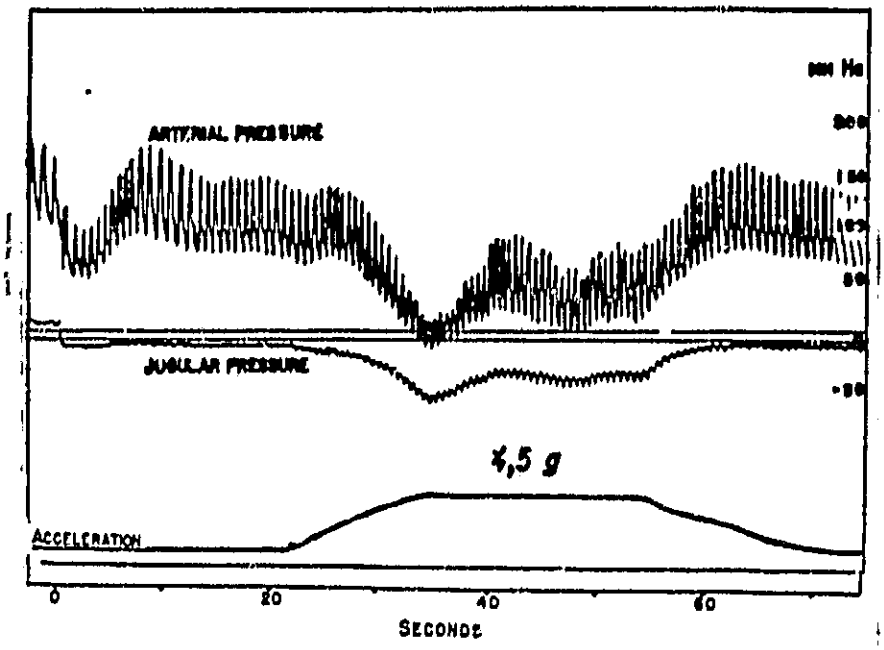


Figure 4: Model response to $+G_z$ acceleration:
Cardiac output and total peripheral resistance

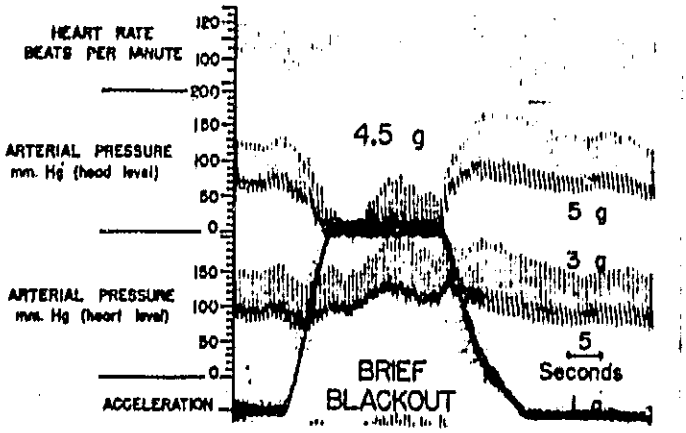
●---● Lindberg and Wood (18)

——— Simulation

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Source:
Gauer (4)



Source:
Lindberg & Wood (18)

Figure 5: Cardiovascular response to headward acceleration of 4.5 g in normal subject from two separate experimental studies

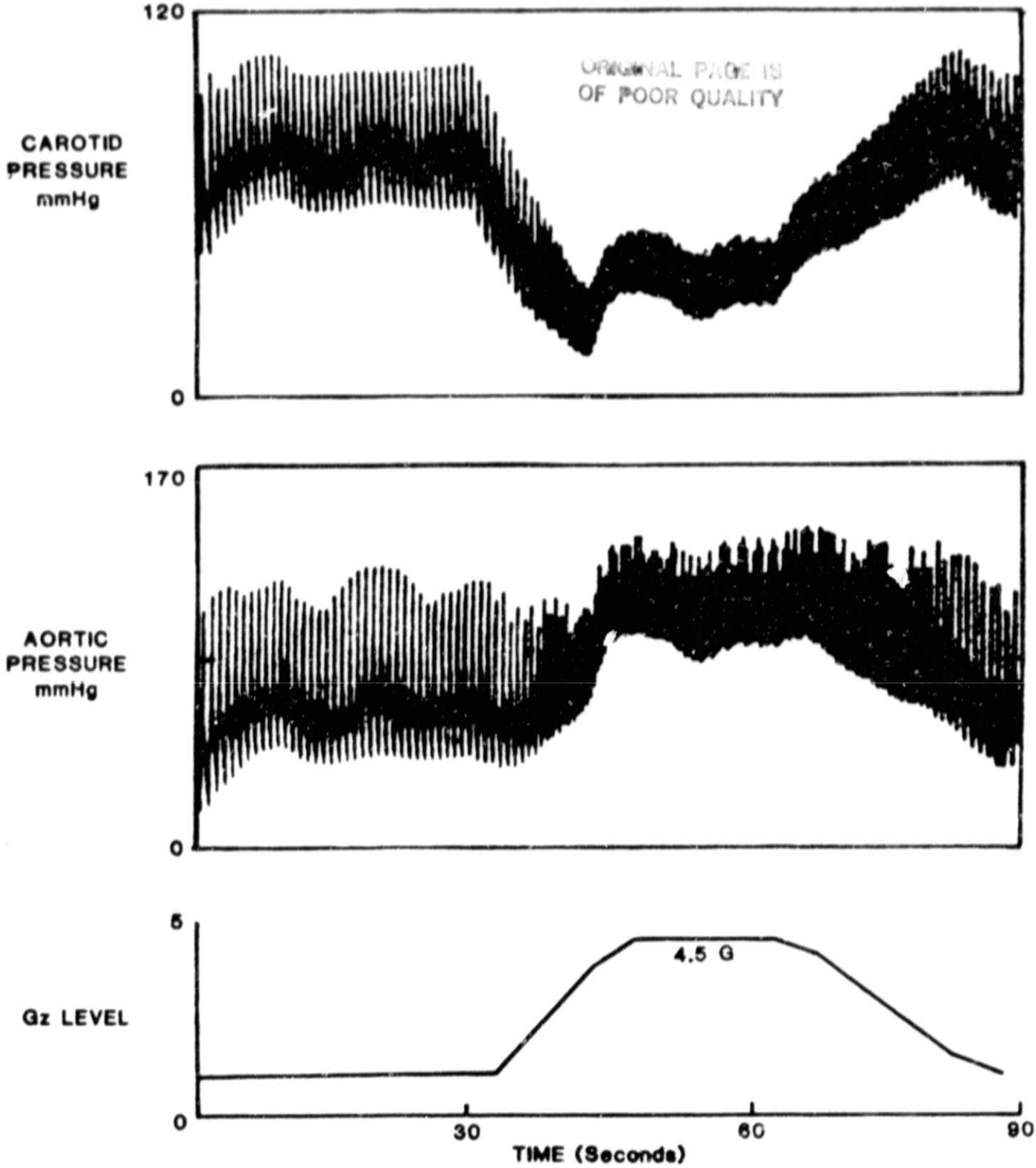


Figure 6: Blood pressure waveforms produced by the model to headward acceleration of 4.5 g

a) Narrowing of the pulse pressure immediately after $+G_z$ onset.

b) Decreases of pressure in the head region and increase of pressure in the region of the heart.

c) A rebound effect at the top of the G-ramp reflecting the reflex action of the baroreceptors.

d) An increase in the waveform frequency during centrifugation reflecting an increase in heart rate.

e) Oscillatory behavior of systolic and diastolic pressures originating from changes in intrathoracic pressure imposed by respiratory cycles (a phenomenon seen more clearly during the control period).

The success of these validation studies, both in the steady-state and dynamic modes, is gratifying particularly in view of the fact that they were accomplished without altering the basic model which was not originally intended to simulate the effects of high g-forces upon the cardiovascular system.

C) SIMULATION OF G-TOLERANCE CURVE

The level of $+G_z$ acceleration that the body can withstand has been studied extensively by Stoll (21) and is graphically illustrated by the classical G-tolerance curve shown in Figure 7 (Curve C). This curve was generated from data collected on human centrifugation studies in which subjects were exposed to increasing $+G_z$ acceleration until they reached blackout as indicated by loss of peripheral and central vision. Thus, the area above the G-Tolerance curve represents the blackout region, the area below the curve being the region of tolerance.

A computer simulation study was performed to assess the ability of the pulsatile cardiovascular model to reproduce the G-Tolerance curve (20). The acceleration rates reported by Stoll were used as driving forces in the model. A point of functional impairment (PFI) was defined to be the point in time at which the systolic lower carotid arterial pressure (SLOC) decreased to a threshold level of 60 mmHg. The effect of introducing a two-second time delay for oxygen transport was also studied. This time delay is the time between SLOC reaching the threshold level and the time at which PFI actually occurs.

Figure 7 depicts the experimental data of Stoll and model-generated curves with and without a time delay for comparison. This validation study confirms that the model will generate a curve of the approximate shape as the curve derived from experimental data, and suggests that a criteria of threshold SLOC of 60 mmHg and a two-second delay yields the best fit to the data. The differences between the experimental and model curves from two to four seconds seem to be due to a lack of rate-sensitive baroreceptors in the model. Note that in curves B and C, the minimum points of G-tolerance (3.0 to 3.6 g) as well as the time durations for the minimum to be reached (10-11 seconds) are similar. Also, a rebound effect appears in the experimental data as well as in the model curve indicating a slightly greater tolerance for more prolonged g-loading. This is probably due to increasing effectiveness of the baroreceptor reflex with time.

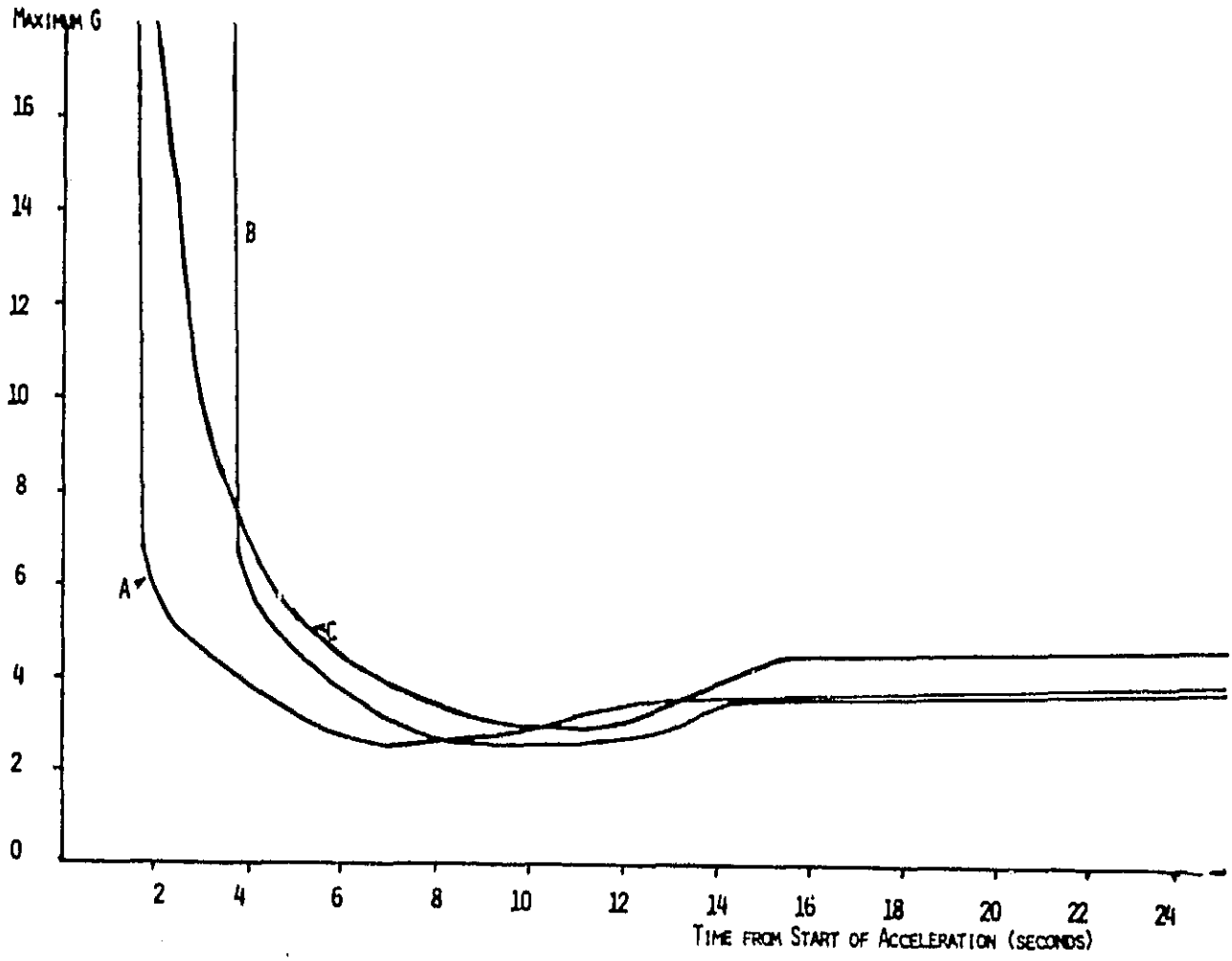


Figure 7: Comparison of experimental and simulated G-tolerance curves

A. Simulated curve with PFI at SLOC of 60 mmHg

B. Simulated curve with PFI at SLOC of 60 mmHg and with compensation for 2-second delay

C. Curve generated from experimental data by Stoll (21)

SIMULATION OF RESPONSE TO BLOOD VOLUME LOSS

In addition to g-forces, a second major factor to consider is the blood volume loss which is present during the reentry maneuver and which has profound effects on cardiovascular responses. The effect of blood loss is of importance because reentry from space flight will take place at a time after the crew is expected to have lost up to 15 percent of total blood volume. Computer simulations of hypogravity, including space flight, water immersion and head-down tilt, have shown a blood volume loss that begins to occur within hours following the onset of stress. These simulations were performed using the Guyton model and are discussed in the Appendix. It was not possible to locate experimental data showing the combined effects of simple blood volume loss (uncomplicated by bed rest or other conditions) and g-forces on cardiovascular parameters. Nevertheless, these effects were simulated using the pulsatile cardiovascular model. The model predictions will have to be verified as experimental data become available.

In this phase of the study, the heart rate and the systolic pressure responses were examined in a parametric fashion at different levels of blood volume loss and g-loads. The systolic carotid pressure is important because this parameter reflects the adequacy of cerebral blood flow. The normal expected blood volume loss in astronauts returning from space flight is in the neighborhood of 8-12 percent. Accordingly, blood volume losses of up to 15 percent were used in the simulation. The results (Figure 8) indicate the following effects:

a) Blood loss combined with $+G_z$ loading results in decreased central blood volume and filling pressures.

b) At any given level of g-force, the model predicts that blood pressures will fall and heart rate will increase as blood loss becomes larger.

c) In most cases, these trends become proportionally more severe and more non-linear at higher levels of $+G_z$.

The non-linearity of the response is evident from these results. Note that at 2 g, the systolic carotid pressure drops approximately 15 percent from its 1-g value when blood volume is normal, while the same decrease is nearly 50 percent when blood volume is reduced by 10 percent. Similarly, the same blood volume loss at 2 g causes the heart rate to increase by 70 percent compared to only 20 percent for no blood volume loss. The clinical significance of these disturbances will be discussed later. The results attest to the exquisite sensitiveness of the cardiovascular system to changes in both acceleration stress and blood volume loss. Furthermore, they suggest that the model has the potential to become a useful tool for predicting physiological states that are difficult to attain in the laboratory.

Having tested the model for combined acceleration stress and blood volume loss, attempts were made to simulate space-flight reentry and to compare the results with the observed responses to actual space flight.

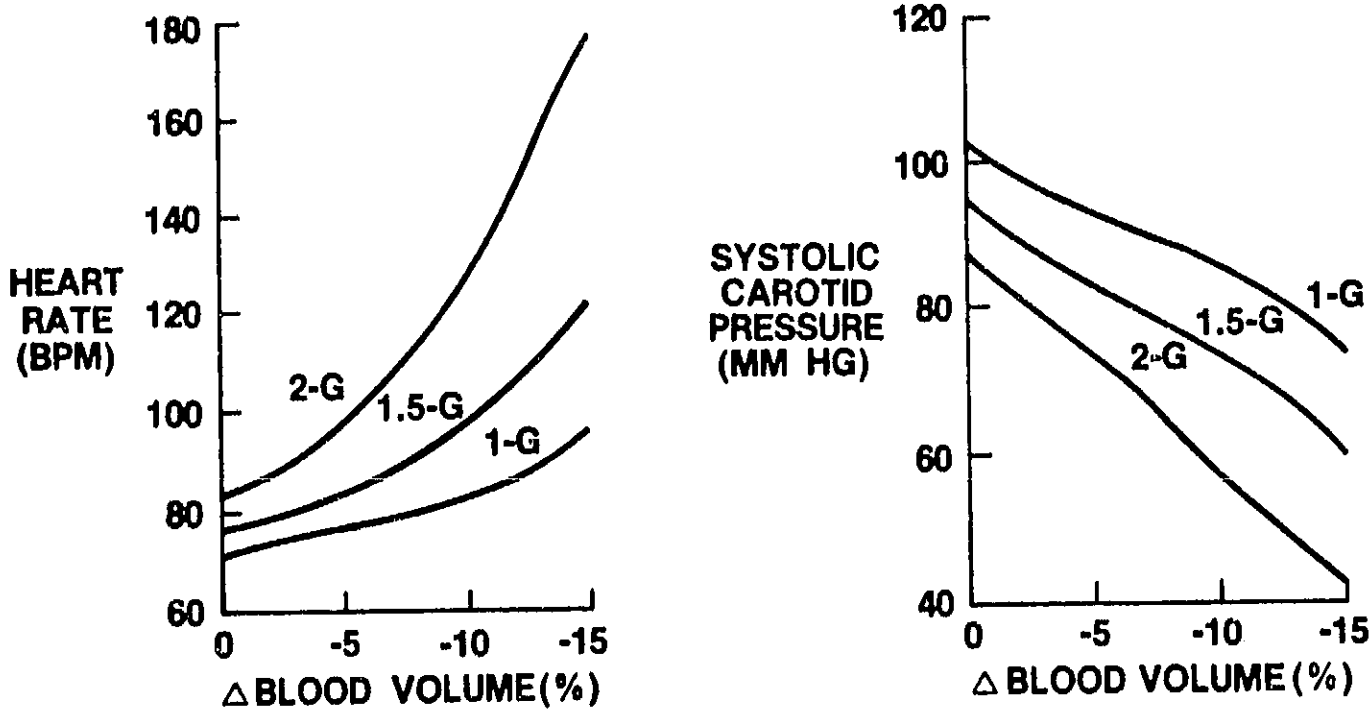


Figure 8: Simulated response to blood volume loss during $+G_z$ acceleration

SIMULATION OF STS-1 REENTRY HEART RATE

A) HEART RATE

Figure 9 illustrates some recorded data from the first Shuttle flight, STS-1 (1). Heart rate was the only physiological variable of significance that was monitored during reentry, a period lasting approximately 25 minutes. The data on heart rate from the two STS-1 crewmembers are shown in the top-half of Figure 9 by dotted and dashed lines. Note the similarity in the responses of the two crewmembers. The data were entered into a data base to facilitate comparison with the simulated heart rate response produced by the pulsatile cardiovasular model.

The curve at the bottom of Figure 9 illustrates the g-force encountered by the STS-1 crew during reentry. It rises from zero-g to a peak, in this case to about 1.6 g, and then returns to one-g at landing. This g-force profile was entered into the model as an input driving function, in the same manner as the ramp acceleration was used to drive the simulation of Figure 6. The effect of blood volume loss, assumed to occur in weightlessness prior to reentry (zero-g hypovolemia) was evaluated for its influence on the reentry response. Simulations were performed at three values of blood volume loss using the same flight g-profile. Therefore, the model's response includes two distinct and separate effects, i.e., a time-varying g-profile and a fixed blood volume loss.

The simulation results are included in Figure 9. The model's sensitivity to blood volume is evident from these results. Overall, a blood volume loss of 8 percent generates a response which approximates most closely the crew's heart rate data. Agreement is poorest during the last 5 minutes or so of flight, although the curve representing 16 percent blood volume loss tracks the data well for this end-of-flight phase. (Blood volume was not measured in these crewmembers.) There are two possible explanations for this discrepancy. First, it is conceivable that the heart rate is reflecting a psychogenic component related to anticipation, excitement, or anxiety that may occur when the crew prepares to take over manual control for landing. This effect has not been accounted for in the model. Equally plausible is the explanation that blood volume losses actually increased during the latter phase of reentry, as suggested by the model, due to excess capillary filtration of plasma into leg tissues. Centrifugation studies reveal that plasma volume losses of 6-12 percent are typical due to this effect (5). The present data, limited as they are, do not permit a resolution of these two explanations.

B) BLOOD PRESSURE

Blood pressures were not monitored during the reentry phase of the flight. The model, however, is capable of predicting blood pressures throughout the system circulation. Some of the results are shown in Figure 10. The g-force profile is the same as the one shown in Figure 9. The center box contains predictions of aortic pressure - the pressure we believe one would find using an indirect blood pressure cuff device. The time-course of this pressure, at any level of blood volume loss studied, does not appear to change significantly during reentry. However, the carotid pressures, those at head level, are predicted to fall to much lower levels and, therefore, could

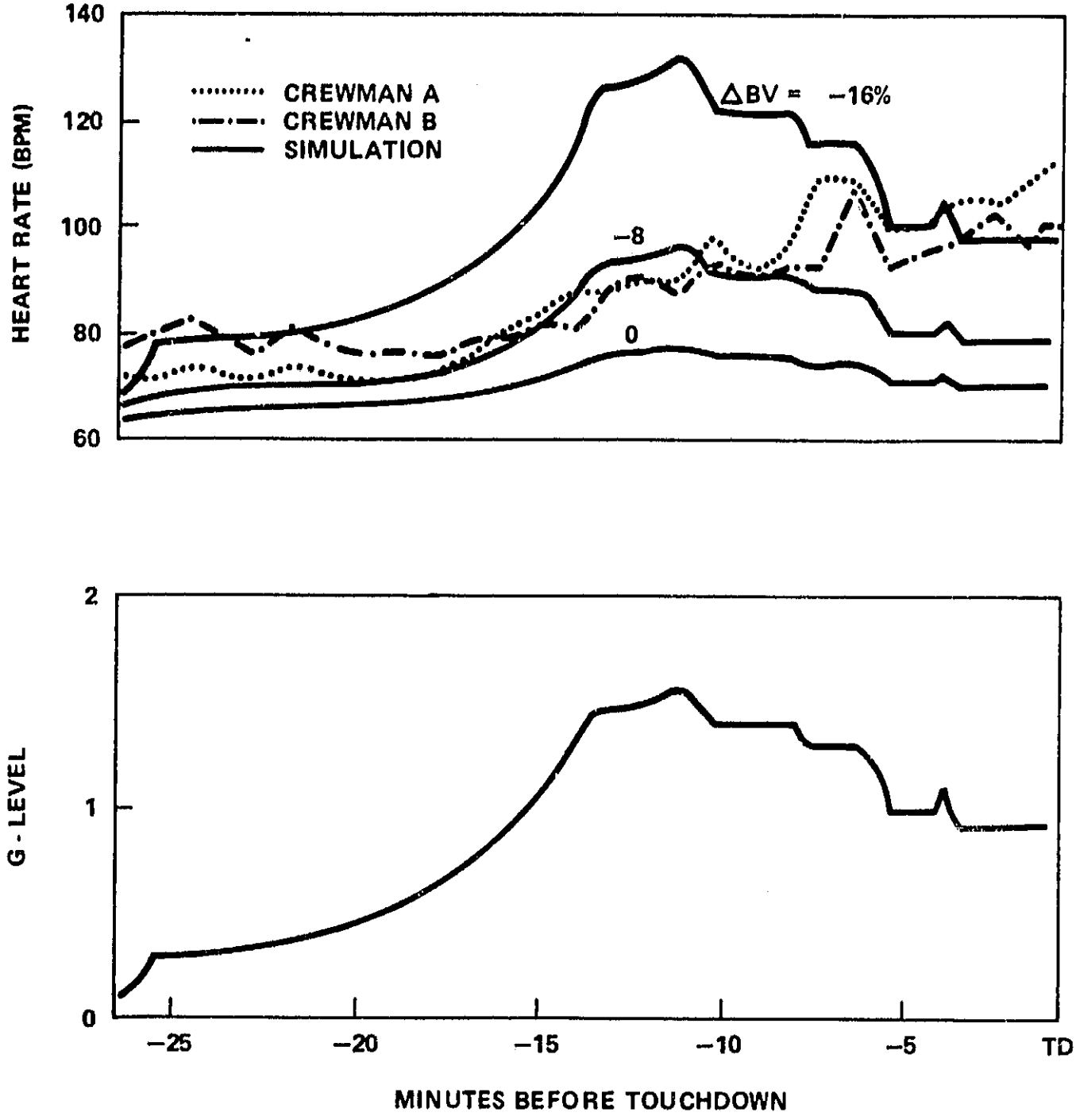


Figure 9: Heart rate response during STS-1 reentry

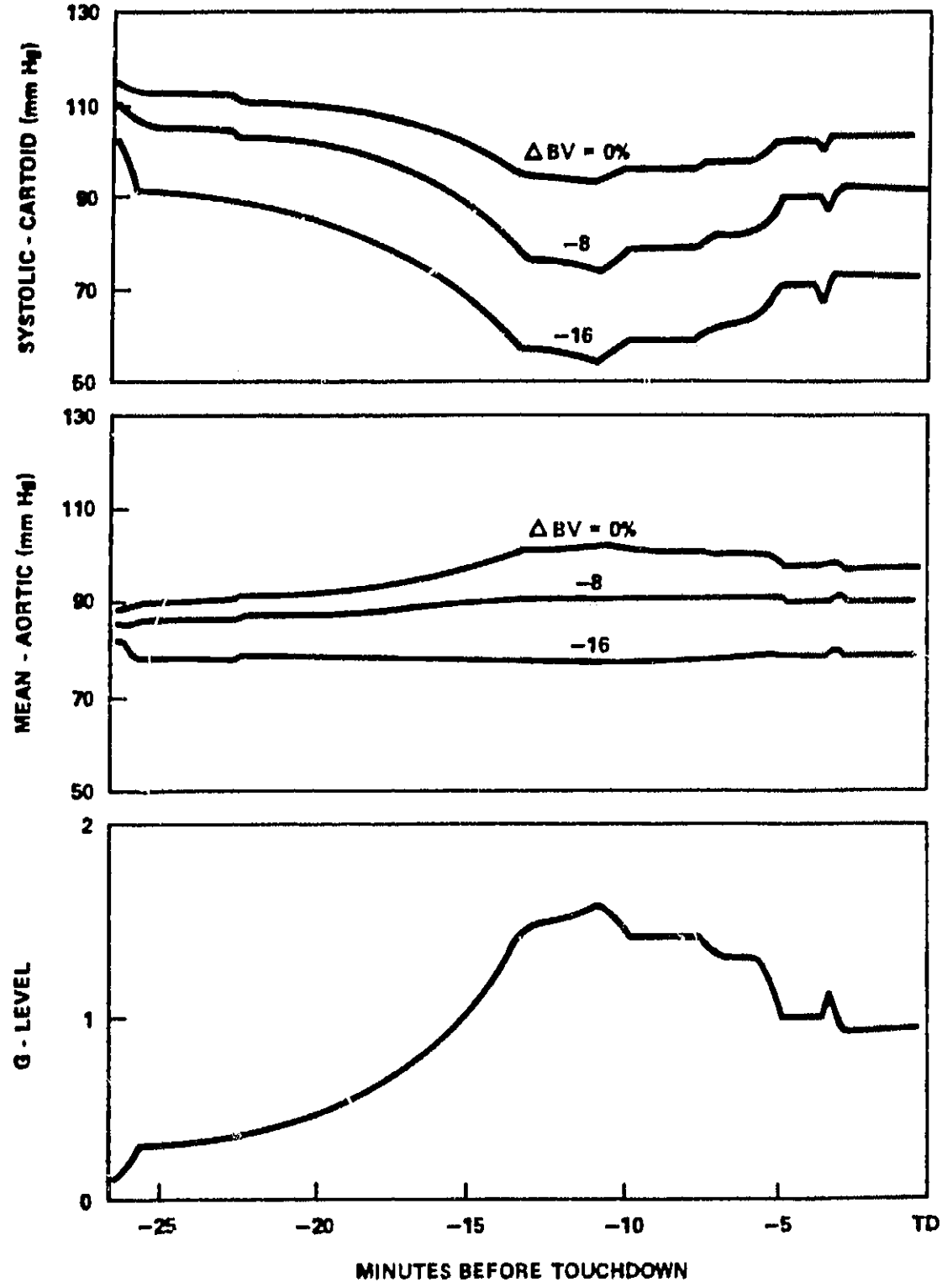


Figure 10: Simulated blood pressure response during STS-1 reentry

effect blood flow to the brain. An attempt was made to assess the clinical significance of these predicted changes. This is discussed in the next section.

CLINICAL SIGNIFICANCE OF SIMULATED RESPONSES

Vision impairment is often used as a criteria of orthostatic intolerance to headward acceleration. The results from one such study is shown in Figure 11 which show the correlation between the systolic arterial pressure recorded at head level during exposure to headward acceleration and the symptoms produced in 18 normal subjects (with normal blood volume) during centrifugation (18). Although there is a wide variation in the data, (i.e., a wide range of pressures associated with the symptom from subject to subject, it is safe to use the highest pressure in each vision group as a conservative estimate of the threshold head pressure for symptoms. Thus, a pressure of 62 mmHg would be the threshold pressure for some signs of vision impairment, and the first signs of blackout may be expected at a pressure of 38 mmHg. These threshold pressures are shown as dashed horizontal lines in Figure 12.

The solid curves in Figure 12 are the same as those in Figure 8. They represent simulation predictions of head-level systolic pressure over a range of $+G_z$ forces and blood volume losses. The pressure decreases non-linearly as g-forces and blood losses increase. When the experimental data (dashed lines) are combined with the predicted responses (solid lines), it appears that if blood volume is not altered from preflight, reentry forces which peak at 1.6 g or even 2.0 g, may be tolerated. However, these same g-stresses, when accompanied by only moderate blood volume losses, may be reason for medical concern. The STS-1 crewmembers who were exposed to peak g-forces of 1.6 g, appear to be in a borderline area if their blood volume loss was between 10 and 15 percent. This may represent a worst case scenario in view of the conservative threshold levels used and the lack of experimental evidence to support the simulated blood volume loss effect, but it seems to warrant closer examination.

PREDICTED RESPONSES OF STS-2 REENTRY

A simulation of STS-2 reentry was conducted between the time of the STS-1 and STS-2 flights. It was known that the crew of STS-2 would experience somewhat higher g-forces than was observed on STS-1. The mathematical model could, therefore, be used to obtain a predictions of the physiological effects of reentry for the crew of STS-2 that had not yet flown. An estimate of the g-forces that would be encountered during STS-2 reentry was available and was used to drive the model similar to the simulation of STS-1 reentry discussed previously. All other aspects of the simulation were similar with the exception, of course, that no crew data were available. The major difference between the STS-1 and STS-2 g-profiles was that transient peak g-levels were expected to be close to 2.0 g in the second mission compared to 1.6 g in the first. The simulation provided estimates of peak heart rates and blood pressures which might be encountered in the STS-2 mission.

The results of the STS-2 reentry simulation are shown in Figure 13. The model predicts that, if blood volume losses are at the same level as for STS-1 (estimated at 10-12 percent) heart rates at peak g-forces will only be slightly higher than those for STS-1 (about 10 beats/min higher). If heart

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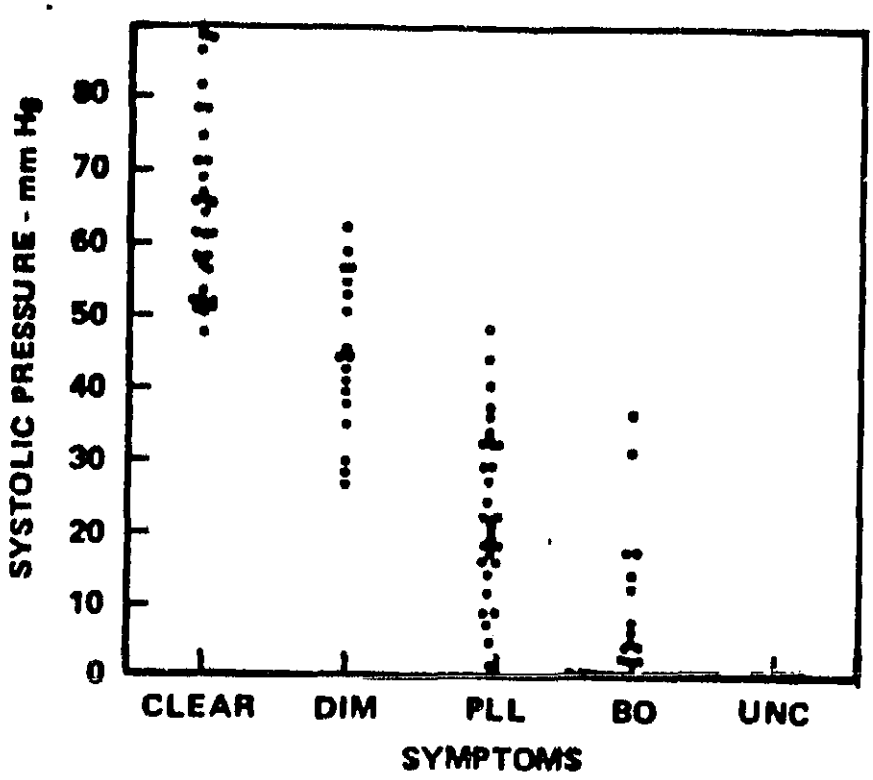


Figure 11: Minimal systolic arterial pressure at head level versus visual symptoms produced during headward acceleration

Source: Lindberg and Wood (18)

Legend:
- CLEAR = NO DETECTABLE VISUAL IMPAIRMENTS
- DIM = PARTIAL IMPAIRMENT OF PERIPHERAL VISION
- PLL = LOSS OF PERIPHERAL VISION
- BO = LOSS OF BOTH PERIPHERAL AND CENTRAL VISION (BLACK OUT)
- UNC = LOSS OF CONSCIOUSNESS

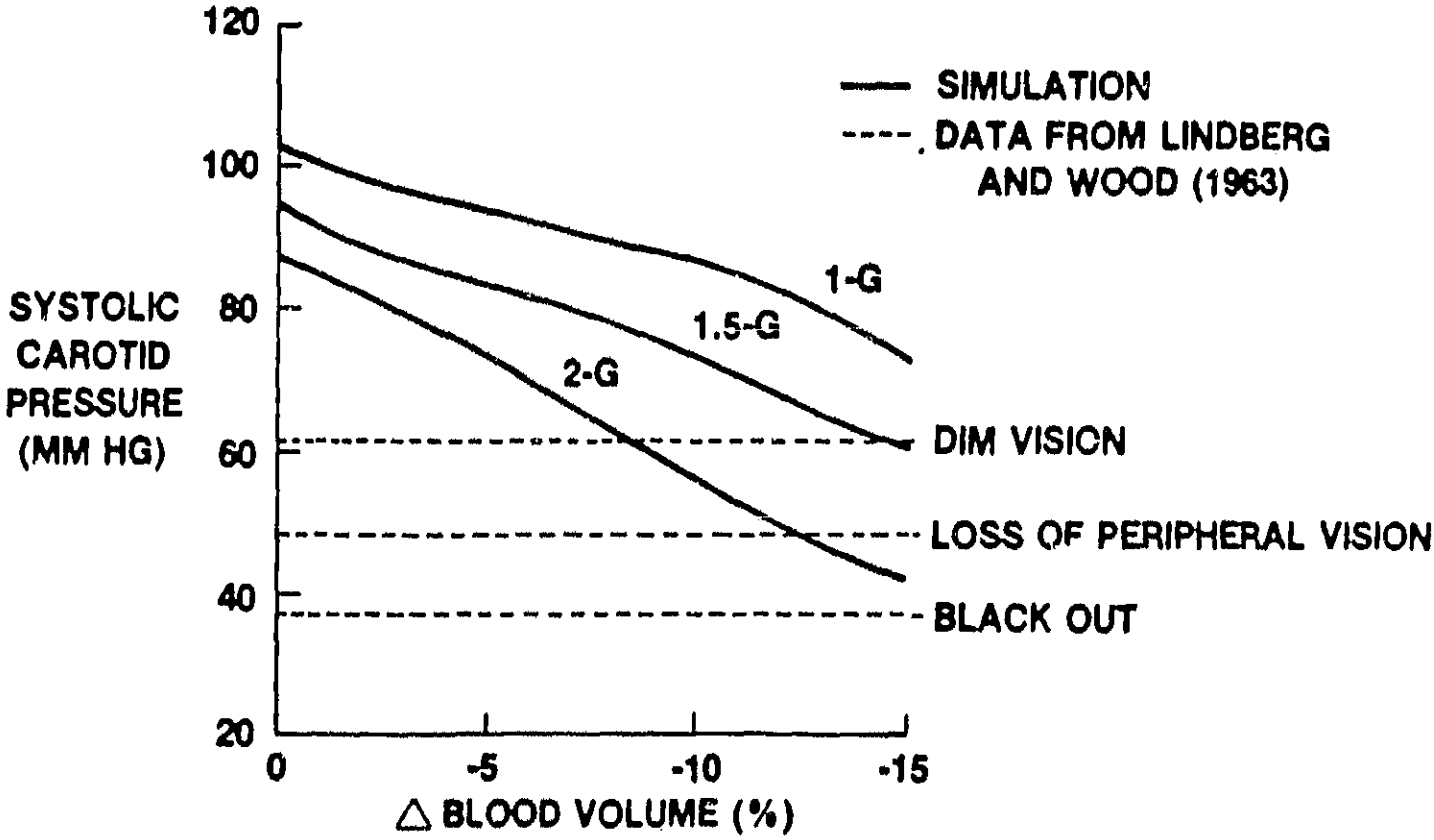


Figure 12: Effect of acceleration stress and altered blood volume on vision

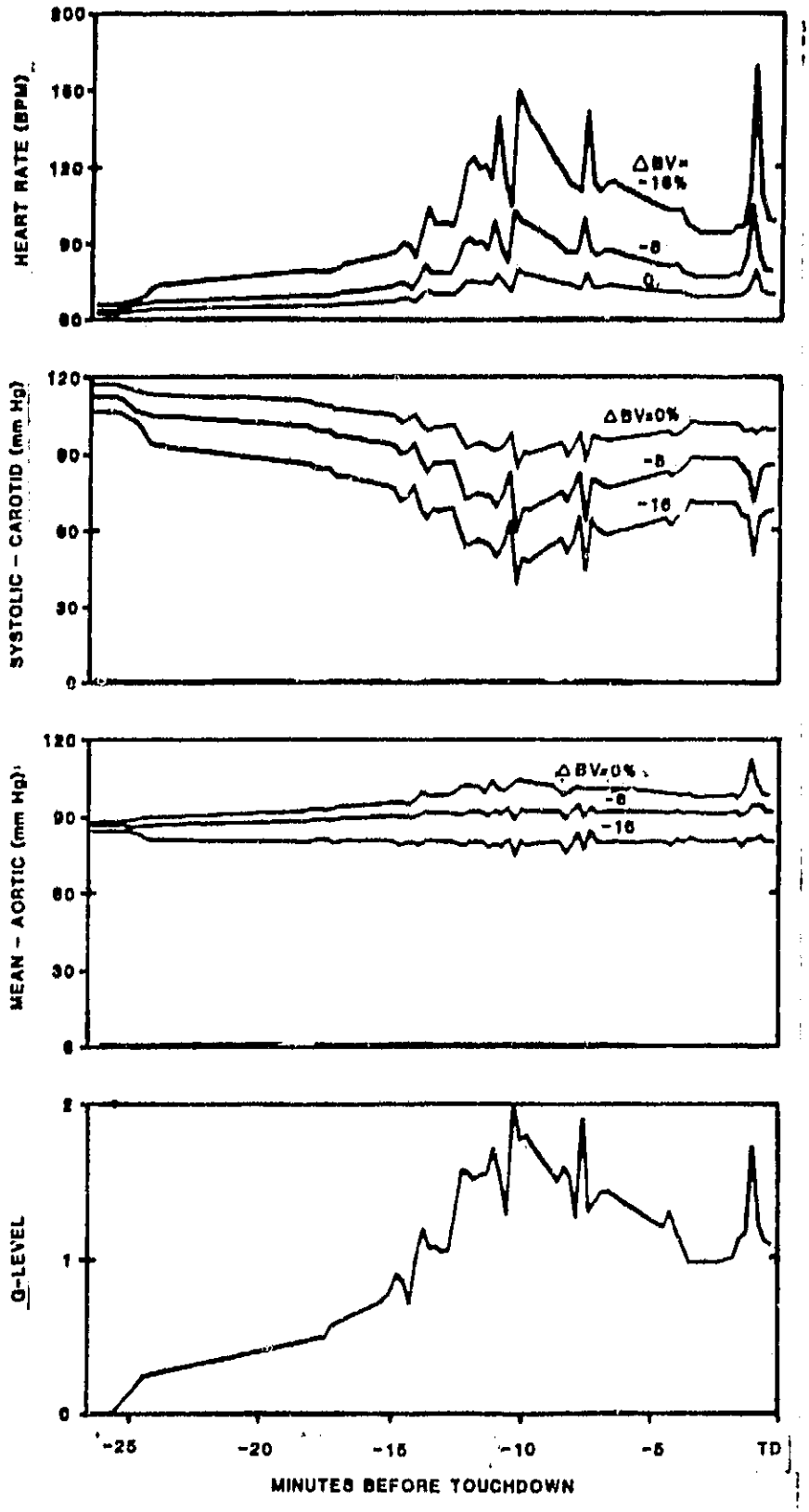


Figure 14: Simulated cardiovascular response to STS-2 reentry

rate is influenced only by blood volume losses, and if blood volume losses of up to 16 percent are experienced, then peak heart rates may reach 150 beats/min. Even if smaller blood volume losses occur of the order of 8 percent occur, we may still expect peak heart rates near the end of the flight to reach about 140 beats/min; i.e., 20 beats/min above the physiological values shown, if the same psychogenic component assumed for the first flight is present for the second flight. (We might add that peak heart rates of the STS-2 crew recorded during reentry were about 150 beats/min which is in accord with this analysis.)

Mean aortic pressures are predicted to be unaltered during reentry. Systolic carotid pressures, however, may dip as low as 60 mmHg for a moderate (8 percent) blood volume loss or 40 mmHg for a more severe (16 percent) blood volume loss. Head pressures this low have been previously associated with vision dimming and loss of peripheral vision as shown in Figures 11 and 12.

SIMULATIONS OF STS-1 CREW STAND TESTS

The biomedical monitoring program of the STS flights include a pre- and postflight stand test administered to each crewmember. At the time this study was conducted the stand-test results from only the STS-1 crew were available. The model we have used to simulate orthostatic tolerance at high-g is capable of simulating orthostasis at one-g as occurs in tilt-table and stand tests. Figure 14 illustrates the responses of the two crewmembers on the left and a range of model-predicted responses on the right. The crew data consists of heart rate and blood pressure range (from systolic to diastolic) during supine (hatched boxes) and during standing (clear boxes).

Simulations were performed by imposing a zero and ninety degree angle of body tilt on the model representing respectively, supine and erect postures, and recording the responses after steady-state was reached (approximately one minute of simulation time). Simulation of the postflight stand test required knowledge of the initial state of the cardiovascular system, presumably altered by exposure to weightlessness. Changes which occurred in the crew as a result of space flight and which resulted in a difference between preflight and postflight tests had to be transcribed to the model, by changing appropriately one or more of the model parameters. Unfortunately, the mechanisms underlying these changes are not well known. It should be noted that the suggestion of causative factors is one of the powerful uses of mathematical models. Two principle mechanisms have tentatively been identified: hypovolemia and abnormal pressure receptor reflex regulation. In these preliminary studies only the hypovolemic effect, i.e., blood volume loss, was evaluated. Thus, on the right side of Figure 14, the model-predicted changes in heart rate and blood pressure are shown for both supine and standing at normal blood volume, corresponding to zero blood volume loss and denoted as "Simulation, Preflight." The predicted results at three different levels of blood volume depletion (5, 10, and 15 percent) are shown, as well. These results assume that blood volume loss alone can account for the differences between pre- and postflight.

With respect to the stand test of crewman "A", the model response which best approximates the postflight response is that which corresponds to 10-15 percent blood volume loss. Note that, the model correctly predicts the characteristics between preflight and postflight results. That is, there is a

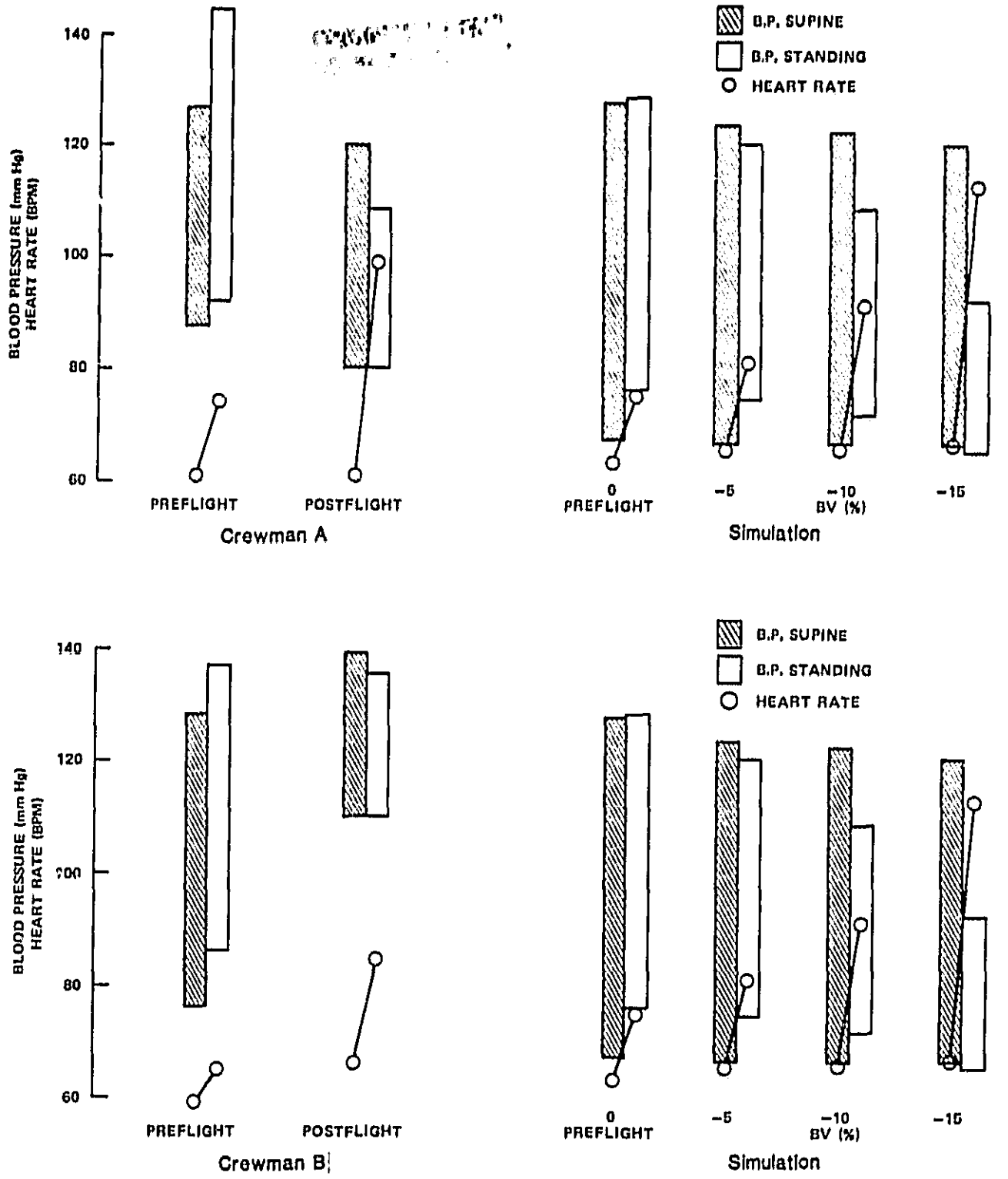


Figure 14: Measured versus simulated responses to stand test. Measurements from STS-1 crewmen are shown on the left. The simulated responses shown on the right are the same in both cases.

narrowing of the pulse pressure, a lowering of the systolic pressure, a flattening of the diastolic pressure, and an increase in postflight heart rate as compared to preflight. The changes are particularly evident in the standing position.

The response of crewman "B" is quite different, however, in one major respect from that of crewman "A"; crewman "A" demonstrates diastolic hypotension (similar to the model responses) while "B" exhibits diastolic hypertension in the postflight test compared to the preflight control. In the light of this discrepancy in data, the model cannot be expected to predict correctly both crewmen's responses. It is probable that an unknown mechanism is activated in one crewman, but not in the other. A useful follow-up simulation study would entail hypothesizing and testing in the model one or more mechanisms (baroreceptor sensitivity, catecholamine release, venous tone, etc.) which could account for the differences in the responses of the two crewmen. These theoretical studies could possibly suggest definitive tests in the crewmen that would be useful in predicting performance.

CONCLUSIONS

This feasibility study has produced the following results:

a) The pulsatile cardiovascular model has been successfully validated over a wide range of $+G_z$ levels for both steady-state and dynamic conditions, with regard to blood pressures, heart rate, and tolerances.

b) Sensitivity studies indicate that blood volume losses, in the range expected under space-flight conditions, will significantly increase the degree of orthostatic intolerance during reentry. These model predictions lack experimental confirmation regarding the specific role of blood volume loss on hemodynamics during +Gz-loading.

c) Comparison of crew heart rate data with model predictions indicates that the slowly increasing heart rates observed in the crew during the 30-minute period prior to touchdown may be best explained by increasing g-forces and that heart rates during peak g-forces may be best explained by a combination of g-force and moderate blood volume loss. During the last 7 minutes prior to touchdown, the high heart rates exhibited by the crew at a time when g-forces were less severe, appear to be due to either a more severe blood volume loss or a psychogenic component that is not included in the model.

d) Simulations of the crew stand test, pre- and postflight, suggest that a moderate blood volume loss occurred. These results are also supported by a model of blood volume regulation (see Appendix) which indicates a 10-15 percent blood volume loss during the first 24 hours of space flight.

e) Correlation of the predicted carotid pressure with previously determined vision impairment symptoms, lead to the following tentative conclusions regarding physiological status:

1. Reentry g-forces which peak at 1.6 g or even 2.0 g may be tolerated very well with a normal blood volume.

2. However, these same g-stresses when accompanied by only moderate blood volume losses (8-16 percent blood loss is consistent with current data) may be reason for medical concern. That is, hemodynamic status is in a region which has been previously associated with vision dimming and loss of peripheral vision.

3. Furthermore, if aspects of cardiovascular deconditioning other than blood volume loss are present, or if blood volume losses are particularly severe (i.e., injury or dehydration), it becomes more likely that vision and performance impairment will occur.

4. Thus, because of the blood volume loss and cardiovascular deconditioning which occur in zero-g, the moderate forces of acceleration encountered during shuttle reentry become more detrimental to physiological function and place the physiological status of the crew near the borderline of some type of impairment.

5. Therefore, it would be prudent to initiate some corrective measures prior to or during reentry (fluid loading, for example) that would increase the margin of safety, or at the very least provide emergency countermeasures (inflation of an anti-g suit) that could be instituted at the first sign of impairment.

FUTURE WORK

This study has demonstrated the feasibility of using mathematical models and computer simulation as a tool for examining and predicting cardiovascular responses to acceleration stress. We feel this work should be continued in the following areas:

a) The state of the cardiovascular system prior to reentry should be defined more precisely. In the current study, the blood volume decrease was the only physiological change considered as a result of space flight. Other changes associated with "cardiovascular deconditioning" and the resulting orthostatic intolerance might include alterations of vascular compliance, resistance elements, and stimuli affecting cardiac pumping ability. Increased understanding of these phenomena would be accomplished by a careful search of the literature concerning bed-rest deconditioning and orthostatic intolerance, as well as mathematical modeling and computer simulation.

b) A second area which should be investigated concerns the analysis of the effectiveness of countermeasures that could be used to protect the Shuttle crew against the orthostatic intolerance exhibited during reentry. Currently the countermeasures of interest include oral fluid and salt loading and the use of an anti-g garment. However, other countermeasures may also be worthy of simulation study, including exercise (during the mission), administration of an anti-diuretic agent along with fluid loading, and valsalva maneuvers. Simulation studies would provide a means to suggest an optimum countermeasure protocol.

c) Certain specific areas were identified in this study as needing more data. The literature should be searched more completely for information related to blood volume effects on orthostatic response, and for the extent of capillary filtration which occurs during high g-loading.

d) The effectiveness of reentry countermeasures can be truly determined only through the subjective and objective evaluation of crew performance during and following Shuttle landing. Available crew data concerning the type and level of countermeasures applied, criteria used to evaluate crew performance during the mission, and physiological assessment after the landing should be reviewed and analyzed in order to:

1) identify orthostatic indices that improve as a result of an applied countermeasure,

2) predict the relative contribution of each countermeasure to overall performance, and

3) recommend further inflight testing designed to clarify both the orthostatic problems and the benefits of the countermeasures.

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APPENDIX

SIMULATION OF BLOOD VOLUME LOSSES DURING WEIGHTLESSNESS

The model of A.C. Guyton (6) for circulatory, fluid and electrolyte regulation has been used for some time to examine blood volume regulation in weightless environments (see Figure A-1). Simulations of water immersion, supine bed rest, and space flight have accurately predicted renal, interstitial, and drinking influences on blood volume losses (10,11,12,14). More recently, head-down tilt studies, performed on human subjects, have suggested that this experimental stress may be a superior one-g analog of weightlessness with regard to fluid regulation (9,15,22). Some of the simulations of head-down tilt are included in this appendix (Figure A-2 and A-3). These simulations have been found to be quite realistic. With regard to blood volume, the model results (confirmed by data) suggest that within 24-48 hours after exposure to zero-g, an initial plasma volume loss of about 15 percent occurs (equivalent to 11 percent blood volume loss). After this time the plasma volume appears to stabilize at this reduced level. Analysis of space flight and bed-rest data indicates that a significant decrease in fluid intake occurs during the first 48 hours. The appearance of frank motion sickness symptoms can reduce fluid balance further. In some cases, the decreased fluid intake alone can explain the observed reductions in total body water and plasma volume. However, there is good reason to believe that if water intake were normal, a loss of plasma and other body fluids would still occur via renal diuresis. That is, a loss of plasma volume and body fluids may be obligatory in space flight. Since fluid intake deprivation augments such losses, it would be prudent to insure that the crew is adequately hydrated prior to reentry maneuvers.

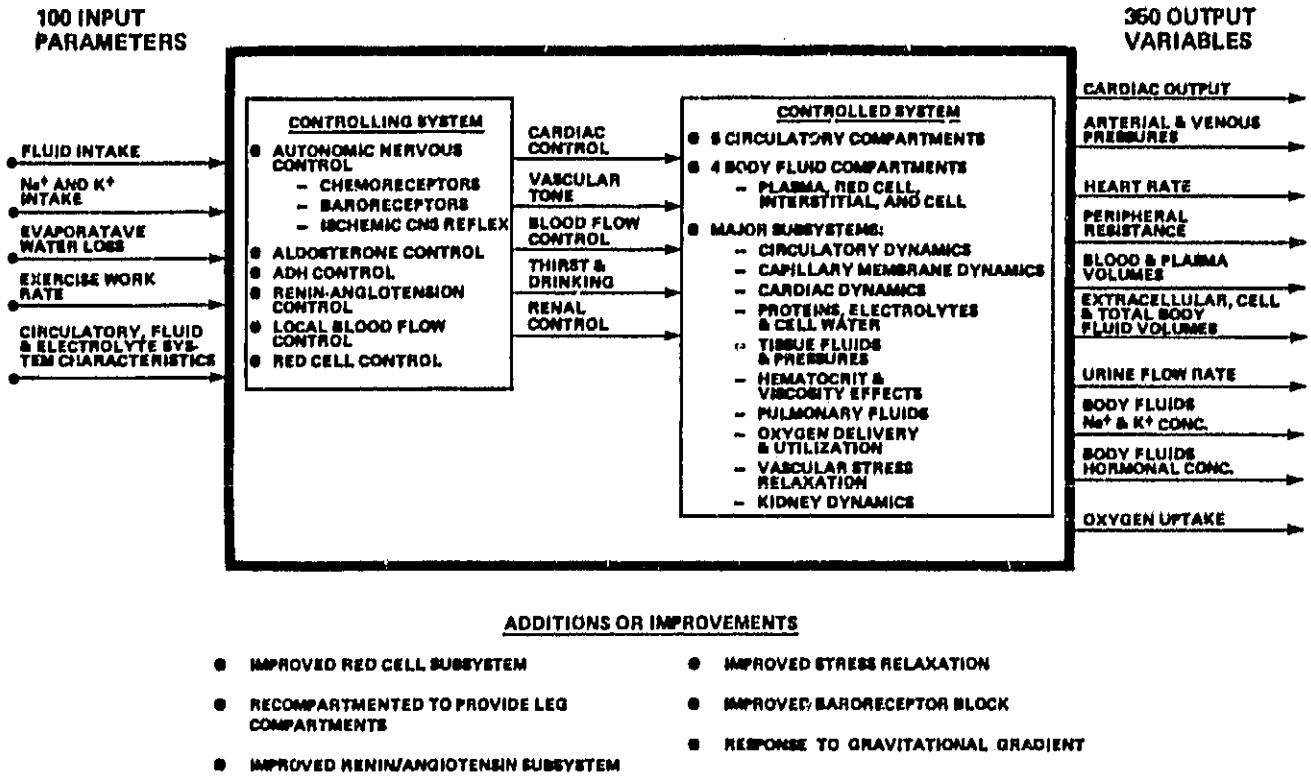


Figure A-1: Model of circulatory dynamics, and fluid and electrolyte balance

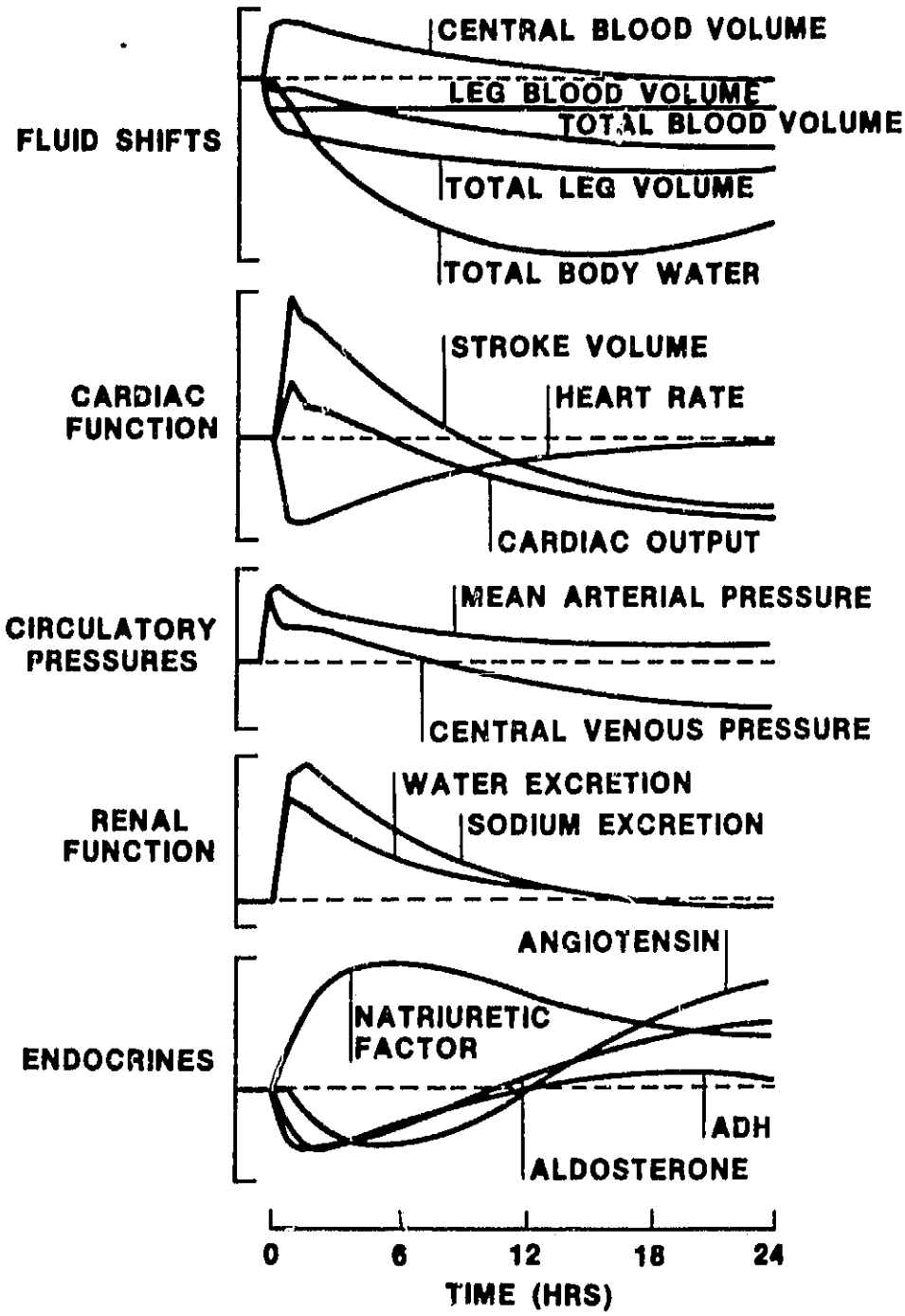


Figure A-2: Simulated circulatory response to head-down tilt (-6^{o})

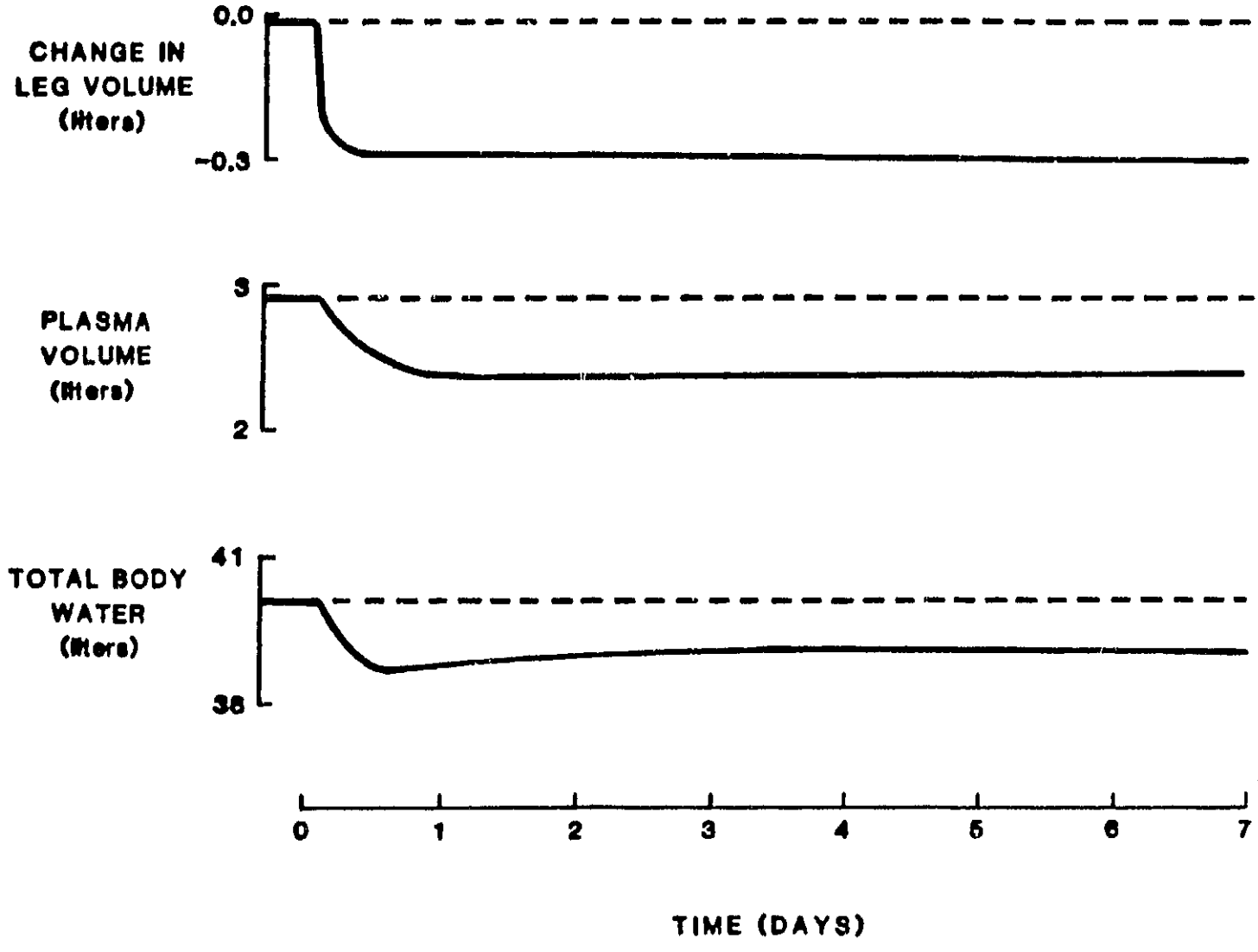


Figure A-3: Simulated response of body fluids to head-down tilt of one-week duration